South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

Vol. 24, No. 44 Cape Town, 4 November 1950 Weekly 2s

A CASE OF RETROPERITONEAL LIPOMA

H. KATZ, CH.M.
Cape Town

Retroperitoneal tumours, by virtue of their size and location, frequently present difficult diagnostic problems. The retroperitoneal lipoma, in particular, is seldom diagnosed pre-operatively.

Case History. A European girl, aged 11 years, complained of increasing adiposity. She felt perfectly well, with no previous illnesses of note. The mother stated that her daughter had always been plump and that for the past six months she had put on a great deal of weight which was most noticeable about the abdomen. She was an active child and her appetite was good. There was no history of abnormal menstruation, nor was there any history of polyuria or polydipsia. There were five other children in the family, and all were perfectly healthy.

On Examination. The child had a healthy appearance, was very fat and weighed 128 lb. The abdomen was very prominent, and the limbs were well developed. The skin was normal in appearance with no hirsutes. The blood picture was within normal limits and the urine revealed no pathological changes. The recti muscles were very well-developed, so that no definite tumour but only a resistance could be felt on the right side of the abdomen. The upper border could not be defined, but the lower border was rounded and extended to a point midway between the umbilicus and the pubis.

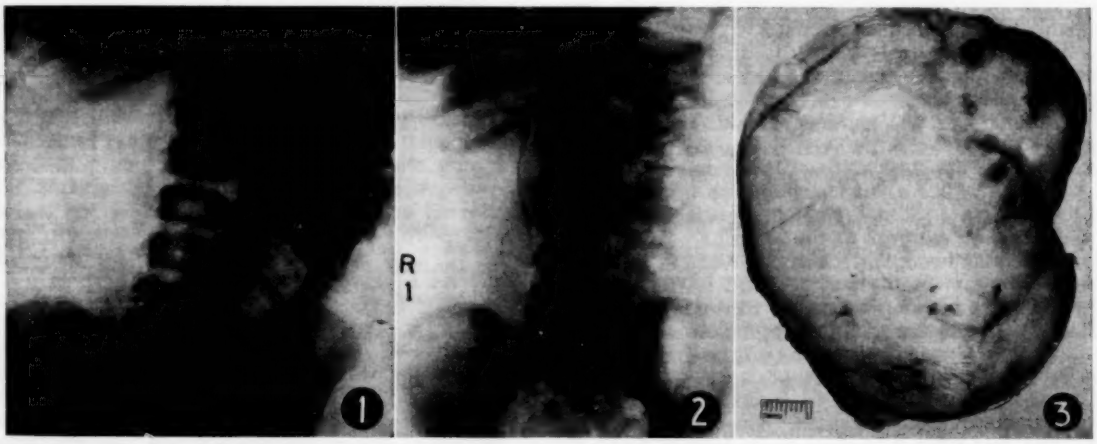


Fig. 1. A barium meal showing the ascending colon and transverse colon lying to the left of the spine in close proximity to descending colon. Note the absence of barium on the right side of abdomen.
Fig. 2. An intravenous pyelogram showing absence of gas shadows on the right side of abdomen. The right kidney is at a higher level than the left.
Fig. 3. The retroperitoneal lipoma soon after removal, weighing 6 lb., and measuring 8½ inches in its longest diameter. Note the well-defined capsule and the absence of any degenerative changes in the tumour.

Medially the tumour reached the midline of abdomen and laterally it appeared to fill the right loin.

Radiological Investigation. X-rays of the skull and limbs did not reveal any abnormalities. The pituitary fossa was normal in size and appearance. The barium meal X-ray, however, yielded some interesting information. The ascending and transverse colon was pushed over to the left side of the abdomen (Fig. 1).

An intravenous pyelogram (Fig. 2) showed the following points:—The gas shadows were evident on the left side of abdomen, with complete absence of gas shadows on the right side. The right kidney was situated at a higher level than the left one, the upper pole of right kidney reaching the upper border of the tenth rib.

Differential Diagnosis. A diagnosis of a large retroperitoneal tumour on the right side of the abdomen pushing the bowel over to the left and the kidney upwards, was considered. The nature of such a retroperitoneal mass, however could not be ascertained.

i. *Suprarenal Cortical Tumour.* Apart from the adiposity, there was nothing else to suggest the presence of such a tumour. There was no evidence of hirsutes and the sexual organs were normal.

ii. *Retroperitoneal Sarcoma.* Such a tumour had to be considered and confirmation was possible only at operation.

iii. *Hydatid Cyst.* The consistency of the tumour and the complete absence of symptoms were well in keeping with this diagnosis. There was also a history of previous residence in a sheep-farming area.

iv. *Wilm's Tumour.* The size of the tumour was compatible with a nephroblastoma or Wilm's tumour, but the age was against such a diagnosis. These tumours are found in young children and have a high degree of malignancy.

Operation. A right paramedian incision extending from costal margin to pubis was made, and a large retroperitoneal lipoma was found. It completely filled the right half of the abdomen, filling up the right loin and displacing the ascending colon, hepatic flexure and all the small bowel over to the left side. The upper pole reached the under surface of the liver and extended as far as the rim of the pelvis below. After incising the posterior parietal peritoneum, the tumour readily shelled out due to complete encapsulation. It derived its nourishment through branches of the right lumbar vessels, which required ligation.

The abdomen was closed in layers after placing a drain in the retroperitoneal space. The tumour weighed 6 lb., and measured 8½ inches in its longest diameter (Fig. 3). There was nothing to suggest malignancy on macroscopic examination. The patient made an uneventful recovery.

Discussion. There are many reports in the literature of large benign retroperitoneal lipomata.[1-6] These tumours appear to be slightly more common in the female, and are usually seen in patients between the ages of 40 and 60 years. It is unusual to find these tumours at so early an age as in the case described above.

Mayo and Dixon [1] found that 80% of these simple retroperitoneal lipomata arose in the abdomen and 20% in the pelvis. The most common site of origin is in the perirenal fat.

The presenting symptom is usually progressive enlargement of the abdomen unassociated with pain. Accurate diagnosis of the type of tumour has rarely been made until exploration has been undertaken. Many of these large benign retroperitoneal lipomata have been removed successfully. The largest tumour on record, weighing 69 lb., was reported by Hirsch and Wells.[4]

These tumours show a marked tendency toward malignant degeneration and recur not infrequently in sarcomatous form even though the original tumour was apparently benign.[5] The tumours exhibit in their malignant status a variety of histologic types, including lipomyxosarcoma, lipofibrosarcoma and true liposarcoma.[6]

They are amenable to surgery and every effort should be made to excise the growth completely. The transperitoneal approach affords the safest access to these tumours. The hazards of surgical excision are considerable and the mortality varies from 20% to 25%.[5]

REFERENCES

1. Mayo, C. H. and Dixon, C. F. (1927): Minnesota Med., **10,** 272.
2. Masson, J. C. and Horgon, E. J. (1931): Surg. Clin. N. Amer., **11,** 105.
3. Andrews, E. (1931): Surg. Clin. N. Amer., **11,** 105.
4. Hirsch, E. F. and Wells, H. L. (1920): Amer. J. Med. Sci., **159,** 356.
5. Catell, R. B. and Warren, K. W. (1947): Surg. Clin. N. Amer., p. 659.
6. McLaughlin, Jr., C. W. and Sharae, J. C. (1938): Surg. Clin. N. Amer., **41,** 512.

ABSTRACTS

Neurosyphilis: Evaluation after Three Years of Treatment with Penicillin alone and with a Combination of Penicillin and Malaria. Curtis, C. *et al.* (1949): Amer. J. Syph. Gonorrh. Vener. Dis., **33,** 527.

Three hundred and eighty patients with various types of neurosyphilis were observed. Treatment consisted of a total dosage of 4,000,000 units of aqueous penicillin, given in doses of 40,000 units every three hours intramuscularly for 100 injections. The fever consisted of tertian malaria, 50 hours or more of fever above 103.5° F. A third of the patients were followed for one year, a third for two years and a third for three or more years after treatment.

The clinical failure rate was 6% with penicillin or with penicillin and malaria combined.

It was found that those treated with penicillin alone responded equally well as those treated with penicillin plus malaria, both with regard to clinical improvement and improvement in the spinal fluid.

Gonococcal Arthritis. Malhotra, S. L. (1950): Indian Med. Gaz., **85,** 187.

The author finds that many cases of gonococcal arthritis do not respond to treatment with penicillin or sulphonamides. He thinks these are caused by mixed prostatic infection mostly with *B. coli* and streptococci. The organisms are identified by culture. He finds hyperpyrexia to be of value in the treatment, but makes no mention of prostatic massage.

South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

VAN DIE REDAKSIE

STREPTOMISIEN EN TERING

Toe gesaghebbendes van 10 lande onlangs in Parys vergader het, het hulle die uitslae vergelyk wat by die streptomisien-behandeling van tuberkuleuse meningitis verkry is. Onder die belangrike verslae wat voorgelê is, was dié afkomstig van lande waar streptomisien-behandelingsentrums gestig is en die gegewens van hierdie sentrums was van buitengewone waarde vir die gekontroleerde waardebepaling van die uitslae wat vermeld is.

Onder die talryke onderwerpe wat bespreek is, was:

i. Die diagnose van tuberkuleuse meningitis;
ii. Die roetes waarlangs kiemvernietigende middels tot die ruimtes van die harsing- en rugmurgvliese toegedien kan word;
iii. Doseerkunde;
iv. Neuro-sjirurgiese operasies;
v. Ondersoek van die harsing- en rugmurgvloeistof;
vi. Die konsentrasie van streptomisien in die liggaamsvloeistowwe;
vii. Elektro-encephalografie;
viii. Ongelukke aan streptomisien te wyte;
ix. Behandeling met kombinasies van geneesmiddels.

Die algemene gevolgtrekking waartoe geraak is, was dat streptomisien slegs gebruik moet word vir die behandeling van tuberkuleuse meningitis, miliêre tuberkulose en primêre besmettings wat op die punt mag wees om algemeen te versprei. Die behandeling by kinders van ander vorms van tering wat vir hierdie geneeskundige behandeling vatbaar is, kan egter oorweeg word waar dit noodsaaklik voorkom.

Ten einde behandeling aan 'n groter aantal gevalle beskikbaar te stel, is daar aan die hand gedoen dat streptomisien-sub-sentrums gestig moet word mits daar verseker word dat 'n hoë standaard van behandeling behandhaaf word en dat dit deur gespesialiseerde personeel toegedien word. Daar is dus by die verskillende regerings aangedring om gebruik te maak van die aanbod van beurse vir die opleiding van spesialiste in streptomisienterapie. Dit sal noue samewerking tussen die sentrums in elke land en tusen dié in verskillende lande tot gevolg hê.

So 'n hoogs gekoördineerde en gestandaardiseerde aanpakking van die probleem sal dit ook makliker maak om inligting oor verskille in die kragtigheid en giftigheid van streptomisien te verkry en sal baie makliker 'n eenvormige metode vir die aantekening van resultate verskaf.

Die gebruik van streptomisien saam met ander kiemvernietigende middels wat reeds aanbeveel is, sal ook doeltreffender ondersoek kan word.

EDITORIAL

STREPTOMYCIN AND TUBERCULOSIS

When experts from 10 countries recently met in Paris, they compared the results obtained in the treatment of tuberculous meningitis with streptomycin. Among the important reports submitted were those which came from countries where Streptomycin Treatment Centres had been established and the data from these Centres were of unusual value in the controlled assessment of the results reported.

Among the numerous topics discussed were:

i. The diagnosis of tuberculous meningitis;
ii. The routes by which antibiotics may be introduced into the meningeal spaces;
iii. Posology;
iv. Neurosurgical operations;
v. Examination of the cerebrospinal fluid;
vi. The concentration of streptomycin in the body fluids;
vii. Electro-encephalography;
viii. Accidents due to streptomycin;
ix. Treatment with combinations of drugs.

The general conclusion arrived at was that streptomycin should be reserved for the treatment of tuberculous meningitis, miliary tuberculosis, and primary infections when generalization may be imminent. The treatment of other forms of tuberculosis in children which are amenable to this therapy could, however, be considered in case of necessity.

In order to make treatment available to a greater number of cases, it was suggested that Streptomycin Sub-Centres should be established provided that care was taken to keep the standard of treatment high and to have it administered by specialized personnel. Governments were therefore urged to avail themselves of the offer of fellowships for the training of specialists in streptomycin therapy. This would result in close collaboration between the Centres in each country and between those in different countries.

Such a highly co-ordinated and standardized attack on the problem would also make it easier to gather information about variations in the potency and toxicity of streptomycin and would provide much more readily a uniform method for recording results.

The combined use of streptomycin and other antibiotics already recommended could also be more effectively explored.

MALIGNANT MALNUTRITION

FRANK WALT, M.R.C.S., L.R.C.P., D.C.H. (ENG.)
LUCY WILLS, M.A. (CANTAB.), M.B., B.S. (LOND.)
Paediatric Department, McCord Zulu Hospital, Durban
and
R. P. NIGHTINGALE
Government Pathological Laboratory, Union Health Department, Durban

The syndrome Kwashiorkor or Malignant Malnutrition was first adequately described by Williams (1933). Later much work on the subject was published by other workers including Trowell (1937-1949), Altmann (1948) and Waterlow (1948). Readers are referred to these papers for a more detailed discussion of the syndrome, as this report deals mainly with the relation of the plasma protein levels and the protein intake to the clinical picture, course and prognosis of the disease.

The syndrome is now generally considered to be a deficiency state most commonly affecting infants and children between the ages of four months and three years. The distinguishing features are anorexia, wasting, oedema, various skin changes and a fatty infiltration of the liver at some stage of the disease. The factors giving rise to the condition are undetermined as yet, but they are associated with a diet consistently low in calories and protein, and relatively high in carbohydrate.

Kwashiorkor occurs in West, East, Central and South Africa and an apparently identical syndrome has been described in many other lands—Mexico (Gil 1934), Gautemala (Ubico and Klee 1938), China (Platt 1945) and Ceylon (Nicholls 1946). Altmann (1948) and Waterlow (1948) point out that the condition is essentially similar to the *Mehlnahrschaden* of Czerny and Keller (1928) which may occur in any infant fed a high starch, low protein diet. The syndrome, therefore, is not characteristic of any one race.

Material. This report is based on studies of 36 consecutive cases admitted to the Children's Wards of this Hospital between 7 February and 19 June 1949. The patients were all Africans living in or near Durban, but they were not entirely representative of the Native population because the parents had to pay for hospitalization and therapy and so made selection on an income basis unavoidable. In spite of this all the families could be classed as poor. Histories are notoriously unreliable amongst Africans but with one exception, the youngest patient, all the children had been breast fed. Symptoms started after weaning, when the infant's food consisted almost entirely of carbohydrate, chiefly mealie meal and kaffir corn porridge, with a minimum of milk which was often absent from the diet. The disease is seasonal, few cases occurring within the winter months of June, July, August and September. Unlike the cases reported by Trowell in his numerous papers and which practically all suffered from some tropical parasitic disease such as malaria or hookworm, none of the cases in the present series had any such infection except a few of the elder patients who had ascariasis.

Diagnosis and Clinical Findings. The diagnosis of the syndrome is clinical and in this series was based upon a combination of some or all of the following *signs*: oedema, which was often so gross as to suggest nephritis; soft, thinned, light-brown or reddish hair; small and scattered areas of hyperpigmentation on the skin 'like blobs of black paint' which often became confluent, then cracked and peeled off to leave a dry or oozing depigmented lesion (these often became infected and led to ulceration); angular stomatitis with ulceration of the mucous membranes of the mouth; a salmon pink colour of the tongue; an enlarged liver and wasting masked by oedema.

Symptoms consisted of marked anorexia with irritability and four to five loose greenish stools per day.

The most frequent combination of signs was oedema, skin changes, stomatitis and lightly coloured hair, but in some cases described as the atrophic form, gross wasting without oedema was the feature and the infant appeared to be in the last stages of starvation. The incidence of these signs and symptoms, together with the sex and mean age are shown in Table 1.

TABLE 1

No. of Cases	*Sex. M.*	*F.*	*Age (Average) in Months*	*Weight (Average)*	*Oedema*
36	24	12	22 (3–72)	16 lb. 15 oz.	32

Liver. Enlarged	*Hyper-Pigmentation*	*Exfoliation and Ulcers*	*Angular Stomatitis*	*Light Brown Hair*	*Diarrhoea*
30	14	18	20	27	19

Therapy and Progress. Treatment was based on the assumption that the syndrome is related to a low-protein, relatively high-carbohydrate diet deficient in calories and on the fact that the liver is known to be grossly fatty in such cases. Excess protein with limitation of carbohydrate and fat was prescribed as 'Maas', a traditional food made for these patients from skimmed cow's milk by the addition of a 'culture' of previously soured cream. The method of preparation is as follows:—

At 4 a.m. 4 oz. of fresh cream are put in a cup which stands in hot water; four hours later 2 oz. of machine-skimmed cow's milk are added to this cream, the surrounding water still being kept warm. Eight hours later two more ounces of skimmed milk are added to the 'culture' and this is allowed to stand for a further 16 hours. Then the contents are added to two gallons of skimmed milk and are stirred in well and left to stand a further 24 hours when it is offered for the first time as 'Maas'.

The quantities given varied from 4-8 oz. every two to three hours depending upon how much it was possible to coax or force the child to take, the appetite being poor on admission. A lot depended upon the

TABLE 2.—SERIAL SERUM PROTEIN VALUES BY THE COPPER SULPHATE METHOD
(*Expressed as Grammes per 100 ml. Serum*)

Time in Days under Treatment

Case No.	0	1—5	6—10	11—15	16—20	21—25	26—30	31—35	36—40	*AS O.P.*
1	4·5		5·6		7·0	7·5				
2	3·9		6·1		7·3			8·0		
3	3·8			6·1		7·4		7·6		
4	3·8			6·8			8·4			8·1 (55d)
5	4·5									
6	5·3		6·6		7·5	7·9		8·2	8·6	8·1 (64d) 7·5 (96d)
7	3·9									
8	4·4									
9	3·5		4·5		6·3	6·1		6·9	7·3	6·9 (44d)
10	4·7									
11	5·7		7·1	7·8		8·2				8·2 (43d)
13	4·5		6·5	7·0		7·5	8·2			
14	5·1									
15	4·0		5·7		6·9	7·7	7·8			
16	3·9		6·1	7·3		6·8				7·3 (59d) 7·4 (97d)
17	5·6									
18	4·2		6·3		7·4			7·8 (OP)		7·2 (62d)
19	4·5		4·4		6·0		6·9		6·7	
20	5·0	5·0								
		5·8	6·6			7·9				
21	4·0	4·6	5·4	6·7		7·3		7·1	7·3	
22	3·7	4·2		5·8	7·1		7·2	7·3		
23	3·5	4·2		6·9	7·6		7·6			
24	5·8		6·4				6·2			
25	5·9		7·1			7·2		7·4		
26	5·0		6·2	6·7						
27	4·2		5·1			7·2	7·6			
28	4·5									
29	4·1		5·1			6·5	6·2		7·3	
30	3·6	4·0	5·7							
		5·4	6·0	6·9	7·2			7·5		
	4·7		6·8				7·5			
32		4·5		5·7	6·9		7·3		7·4	7·6 (45d)
33	4·0		6·1							
34	4·5			5·7	6·9	7·3		7·4	7·6	
36	3·5	4·0	5·6							
		4·9	5·9	6·6		6·8	6·9			
37	4·2	4·0								
		4·5								
		5·0				6·2	6·6			
38	4·1	4·5	5·6							
39	4·7	5·6	6·4							
Frequency	36	15	25	13	12	16	13	10	7	
Mean	4·4	4·68	5·96	6·61	7·0	7·21	7·26	7·52	7·45	
Range	3·5	4·0	4·4	5·7	6·0	6·1	6·2	6·9	6·7	
	to	to	to	to	to	to	to	to	to	
	5·9	5·8	7·1	7·8	7·6	8·2	8·4	8·2	8·6	
S.D...	0·67	0·8	0·84	0·5	0·47	0·79	0·78	0·41	0·58	

Standard Deviation (All values Total Protein)=0·44.

patience and skill of the nursing staff in getting the patients to take a satisfactory amount. The treatment was thus based upon frequent forced feeds. Appetite improved rapidly so that 8 oz. feeds were taken seven times daily in the majority of cases by the third or fourth day. No other food or medicine was given during the first five to seven days with a few exceptions to be discussed later. After seven days of this regime a teaspoonful of skimmed milk powder was added to each feed and this was continued for a further five to seven days when mixed feeding was introduced in all but the youngest infants. The child was generally on a full ward diet complemented with 'Maas' within three to four weeks of admission.

The protein intake on this regime was high. The 'Maas' contained 3.3% protein and yielded approximately 35 calories per 100 gm. On the routine treatment this would mean an intake of 42 to 56 oz. of 'Maas' yielding 40 to 50 gm. of protein (2.5 to 3.0 gm. protein per lb. body weight) and 420 to 560 calories (24.5 to 33.0 calories per lb. body weight) per day.

Clinical improvement was not apparent until between the third and sixth day, when the oedema would start to subside, often being heralded by a marked diuresis. A child weighing 25 to 26 lb. might lose as much as 2½ lb. in weight in the first week of treatment, the oedema disappearing to reveal a very thin emaciated child. Further progress was usually rapid with a gain in weight, loss of irritability and healing of the mouth and skin lesions. The depigmented hair alone remained as a sign of the syndrome and took a long time to recover its normal texture and colour.

Two children critically ill, one being severely anaemic, were given blood transfusions and other supportive treatment. One died and the second was removed by his parents the day following transfusion. Another child with the atrophic form of the syndrome received 1,300 c.c. glucose-saline intravenously over two days and then the routine treatment complemented with oral Hepatex and vitamin C 150 mg. daily. This patient, though critically ill, made a spectacular recovery. The youngest patient, a three-months' old artificially-fed baby with oedema, enlarged liver, skin changes and a moderate anemia was given 'Maas' and a blood transfusion and made an excellent recovery.

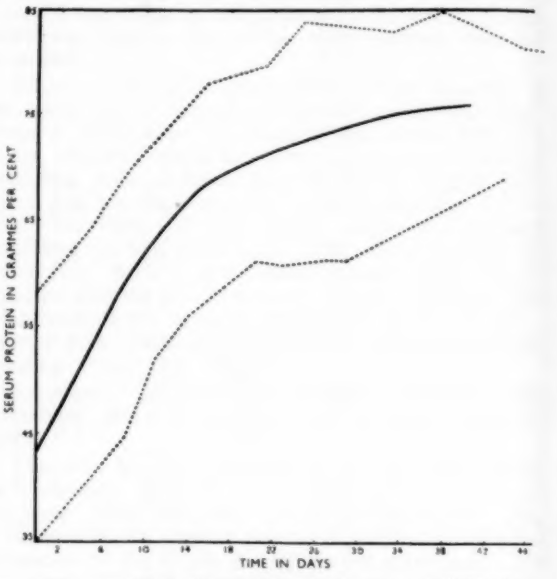
Four deaths occurred in the series (Cases 7, 8, 10 and 17). Three died within 24 hours of admission apparently from liver failure, and the fourth case nine days after admission. The latter child had severe diarrhoea and though oedematous at first, became dehydrated later and failed to respond to therapy despite transfusion of blood, saline and Amigen and died of a terminal lung condition. These findings give a total death rate of 11%; of the six cases in the atrophic stage two died both within 24 hours of admission, giving a death rate of 33.3%. Altmann (1948) excludes from his calculations of death rates all cases dying within 24 hours of admission; on a similar basis the death rate in the present series would have been 2.8% as compared with Altmann's rate of 20% and Gillman *et al.* (1945b) of 6%.

LABORATORY DATA

The investigations carried out were not extensive owing to the limited pathological facilities at the hospital, but it was possible to make fairly complete haematological examinations and serial protein estimations by the copper sulphate method. The protein estimations were checked and the albumin globulin ratio determined by Greenberg's method (1924).

The copper sulphate estimations were all done by one observer (L. W.) who previously in association with Marrock and Hoch of the London Hospital (unpublished data) had found that with a standard technique it is possible to get very good agreement between the results obtained by this method and those obtained by the Micro-Kjeldahl method. She is confident that the error in the present series is not unduly large and that the serial figures are of real value.

Serum Protein. The figures obtained by the copper sulphate method are shown in Table 2 and on Chart I.



Mean Serum Protein in gm. per 100 ml.
Maximum and Minimum Serum Protein Levels.

Fig. 1.

The factor used for the calculation of total protein was P = 364 X (S.G., 1006). On admission the mean serum protein value was 4.4 gm. per 100 ml., range 3.5-5.6 mg. per 100 ml. The initial value was 5 gm. per 100 ml. or over in seven cases some of which had gross oedema, others being atrophic cases without oedema. The rise in the protein level was very rapid starting on the second or third day of treatment, the mean value having risen to 7.0 gm. per 100 ml. by the 16th to 20th day and to 7.5 gm. per 100 ml. by the 31st to 35th day. In one case only had the serum protein value

not risen by the eighth day of treatment, and in only six cases was there even a temporary check in the steady rise while the patient was in hospital. The level in some cases rose to over 8 gm. per 100 ml. and remained high for some considerable time before falling to more normal levels.

Duplicate estimations by the two methods showed fair agreement. The mean difference in 59 duplicate estimations was 0.3 gm. per 100 ml., which gives a mean error of 6.2%. The range of difference was, however, great, from 0.0 to 0.8 gm. per 100 ml., the difference being more than 0.5 gm. per 100 ml. in 16 estimations. Serial estimations by the two methods showed good agreement; for example in one case the figures in grammes per 100 ml. were 4.0 and 4.1; 5.7 and 6.0; 6.9 and 7.2; 7.7 and 8.2; and 7.8 and 8.1 for the copper sulphate and Greenberg's method respectively. Dr. W. J. M. Holman of the S.A.I.M.R., Johannesburg, very kindly checked the total protein figures in two samples by the Macro-Kjeldahl method. The errors were −5.5% and +4.3% for the Greenberg estimations. No satisfactory check of the method of estimating albumin was obtained, but our figures agreed with those of Altmann (1948) and Waterlow (1948) in showing that the reduction in the total plasma protein was largely due to a reduction in the albumin fraction and that the increase in treatment was due to regeneration of albumin. The globulin values were within normal limits.

It was not possible to get a control series of healthy children, but in seven cases in the same group admitted to the wards with minor ailments and apparently otherwise healthy, the mean total serum protein value was 7.1 gm. per 100 ml. (range 6.5 to 7.6 gm. per 100 ml.) all the values except one being over 7.0 gm. per 100 ml.

Haematological Data. The findings are shown in Table 3. There was a moderate, slightly macrocytic, normochromic anaemia in the majority of cases on admission, but in three cases, all fatal, the anaemia was very severe, the red cell count being 1,183,000, 1,276,000 and 1,675,000 per c.mm. on admission respectively. None of the cases had a megaloblastic anaemia. On treatment the packed cell volume, red cell count and haemoglobin level fell to rise again to their original level and higher by the end of the period in hospital. These findings agree with those of Altmann and Murray (1948) except that in our series no hypochromic or megaloblastic anaemias occurred.

Other Investigations. The urine was examined in every case. A trace of albumin was found in one severely anaemic case, otherwise no abnormalities were discovered.

The serum bilirubin was increased in three cases only, two of which died within 24 hours of admission with grossly enlarged and fatty livers. The third case was only very slightly jaundiced and this jaundice cleared in a few days on treatment.

Post Mortem Findings. Limited autopsies were performed on all four cases that died; two of these (Cases 7 and 8) being of the oedematous and two (Cases 10 and 17) of the atrophic type. The former had generalized oedema, Case 7 having in addition free fluid in all the serous cavities. Though both infants were under two years of age, the liver weighed 1 lb. and 1 lb. 2 oz. respectively, and death appeared to be due to liver failure, though both cases had signs of commencing bronchopneumonia. The livers were grossly fatty, the cells containing large fat globules, compressing the cellular structure so that the normal lobular arrangement could not be defined. The fatty lesion was distributed throughout the organ, except the tissues of the portal tract. There was no appreciable condensation or increase of the reticulum fibrils. The pancreas did not show any pathological changes. The kidney in Case 7 which had been severely anaemic and had had a trace of albumin in the urine showed advanced cloudy swelling of the tubules.

The two atrophic cases (10 and 17) were both aged two years, were grossly underweight with brownish

TABLE 3: MEAN FIGURES FROM BLOOD COUNTS ON 36 CASES.

Mean Time in Hospital (Days)		*Red Blood Cells (Millions per c.mm.)*	*Hb* gm%	*P.C.V.* %	*M.C.V.* μ^3	*M.C.H.C.* %	*Bone Marrow*
			On Admission				
0	Frequency	3·3	35	30	29	29	Active normoblastic reaction: no megoloblasts seen.
	Mean	3·27	10·1	32·6	95·7	31·3	
	Range	1·2—4·9	3·5—12·9	11·0—43·0	80—129	29—36	
	S.D.	1·29	2·13	6·49	11·27	1·41	
			Lowest Count				
15	Frequency	21	22	18	18	18	
	Mean	3·15	9·4	28·9	94·4	32·4	
	Range	1·5—4·2	4·4—11·3	13·0—36·0	82—112	29—35	
	S.D.	0·65	1·49	5·29	8·05	1·41	
			On Discharge				
31	Frequency	25	25	21	21	21	
	Mean	3·75	10·9	34·5	91·9	31·9	
	Range	3·2—4·5	9·6—13·0	30·0—41·5	79—103	29—35	
	S.D.	0·49	0·74	3·09	9·38	1·73	

S.D.—Standard Deviation.

hair and only slight skin changes except for a large bed sore in one child. Both were severely anaemic. The post mortem findings in Case 10 were not significant except for a terminal bronchopneumonia and a small fatty liver, weighing only 8 oz., half the weight of those of the oedematous cases. On section the liver felt tough and macroscopically suggested early cirrhosis, but microscopically there did not seem to be any increase or thickening of the reticulum fibrils of the lobules. There was a diffuse deposit of fat throughout the liver affecting most of the liver cells and granules giving the colour reaction of iron were demonstrable in some of the cells. The second atrophic case (17) was found to be suffering from a tuberculous adenitis of the tracheo-bronchial glands and of the glands at the head of the pancreas. There were left-sided pleural adhesions and a lobar pneumonia. The liver size resembled that seen in the oedematous cases. The cells contained fat globules of varying size. There was some increase and condensation of the stroma of the pancreas and degeneration of the tubules of the kidney.

A further autopsy was performed on a four-year-old boy, admitted for gross ascites and an enlarged liver, but with no oedema and no skin or hair changes. Only a very limited autopsy was allowed, but this showed that the patient had died from haemorrhage into the stomach, probably from a ruptured varicose vein of the oesophagus. The liver was hard with a rough surface; the cut surface was fibrous with islands of bile-stained liver tissue between fibrous bands. Microscopically, the appearance was that of cirrhosis with gross distortion of the liver lobules, diffuse cellular infiltration of the fibrous bands, with well-marked proliferation of the bile ducts. Most of the liver cells contained numerous fat globules and these were also present in the lining epithelium of the bile ducts. Iron was not demonstrated in the liver cells. There was a well marked increase in the reticulum fibrils of the liver. The spleen showed enlargement but no other lesions were noted.

DISCUSSION

The syndrome described above is essentially the same as the one described by Altmann (1948) occurring among infants and children in Johannesburg and, except for the complicating tropical diseases, as that described by Trowell (1937-49) in Uganda. Lesions of the skin and mucous membranes are present, often dominate the picture and are important for the clinical diagnosis of the syndrome. In our opinion, however, the fundamental lesions are wasting, fatty infiltration of the liver, and oedema associated with low serum proteins. It was not possible in our series to demonstrate the presence of fatty changes in the liver by biopsy, but in all autopsies the characteristic fatty changes were found and there is no reason to doubt that they were always present.

It is difficult to determine the aetiology of the oedema that is so constant a finding at some stage of this disease and it seems probable that more than one factor may be involved, the low serum albumin level being an important one, though qualitative changes in the serum proteins, salt balance, kidney efficiency, and alterations in the capillary permeability may also play a part. In our series and in most other reported cases a low serum protein appears to be of the greatest importance. The constant history of a diet grossly deficient in protein, the low serum protein (particularly the albumin fraction) and the correlation between the increase in the blood protein level and the decrease in oedema all indicate the importance of protein deficiency in the production of the oedema. The highest initial total serum protein figures obtained in our series, 5.8 gm. per 100 ml. and 5.9 gm. per 100 ml., are both below the accepted lower level of normal (6.0-8.0 gm. per 100 ml.); these values were found in two atrophic cases with only minimal oedema but with enlarged livers. The bulk of the cases had markedly reduced serum proteins, the level corresponding roughly with the extent of the oedema; cases with gross oedema usually had a total serum protein of about 4.0 gm. per 100 ml., while those with slight oedema a value of about 5.0 gm. per 100 ml.

The effect of treatment with a high protein diet in the form of 'Maas' had an immediate effect on the serum protein, which rose very rapidly, an increase often showing by the second day of treatment (Chart I). The rapidity of the increase was greater than that reported by other workers; for example, in Altmann's (1948) series of very similar cases, also treated with a sour milk formula, the mean serum protein value before treatment was approximately the same as ours (4.2 gm. per 100 ml. compared with our value of 4.4 gm. per 100 ml.), yet by the 16th day the mean value was only 5.5 gm. per 100 ml. as compared with our value of 7.0 gm. per 100 ml., a value which his cases only reached after 33 days' treatment, when our cases had a mean value of 7.5 gm. per 100 ml. In no case reported by Altmann did the serum protein rise above 8.0 gm. per 100 ml. and this value was only reached once, whereas four of the cases in our series had values of 8.0 gm. per 100 ml. and upwards and these values might be maintained for some considerable time (Chart I and Table 2). It would seem permissible to conclude that the quicker and greater rise in our cases was due to the very large amounts of protein given (2.5 to 3.0 gm. protein per lb. of body weight) in comparison with Altmann's diet which yielded about 1.6 gm. protein per lb. of body weight. The two diets yielded approximately the same number of calories, extra calories being provided in Altmann's formula by the addition of dextri-maltose; our formula had no added carbohydrate. Waterlow (1948) also gave on the whole smaller amounts of protein and had slower and lesser increases in the serum protein levels than were found in our series. Without a far bigger series treated with 'Maas' and also a control series treated with smaller amounts of protein it is impossible to judge whether the low death rate in our series was related to the treatment or a chance finding.

It has been shown that the cases on admission were moderately anaemic and that, as other workers have found, this anaemia increased when treatment was instigated, the lowest level being reached on an average on the 15th day of treatment and thereafter the anaemia decreased until on discharge the original level of the

erythrocyte count, haemoglobin and packed cell volume percentages had been reached again or surpassed. During the same period the serum protein had been increasing steadily. It was impossible to estimate the blood and plasma volumes in our cases. However, Rossiter (1946) and Walters and others (1947) reported in returned Indian prisoners of war, a syndrome associated with wasting, oedema with gross hypo-proteinaemia, with or without signs of vitamin B complex deficiency, but with a normochromic macrocytic anaemia, in fact a syndrome very similar to that of malignant malnutrition in infants. These workers gave a high-calorie, high-protein, high-vitamin diet and found that there was at first a marked increase in both blood and plasma volumes which accounted for an apparent increase in anaemia though the actual amount of circulating haemoglobin was increased; at the same time as the anaemia apparently increased the plasma proteins increased so rapidly that the percentage values either remained at the same level or increased. This work suggests that similar changes in blood and plasma volumes account for our findings. Rossiter and co-workers found, as we did, that in certain cases the plasma protein values might reach values above normal, which gradually returned to normal with continued treatment.

The rapid and often spectacular improvement in the children's condition suggests that these large amounts of protein are not only well tolerated even by severely ill children, but are more effective than smaller quantities. Only a few children could be followed as out-patients; but these, the children of intelligent mothers who followed instructions about diet, had maintained their progress for periods up to 96 and 97 days. The relapses so commonly reported by other workers in the City, may be rather of the nature of another attack than of a true relapse, due to the child's returning to the very environment and diet that produced the first attack. Field studies and long follow-ups can alone elucidate not only the etiology of malignant malnutrition, but also the relationship (if any) between this condition and cirrhosis of the liver in children. Whatever the etiological factors may finally prove to be, there is no doubt that prevention and cure lie in the fields of economics and education. Poverty and ignorance are the basic factors producing this scourge of infancy, and the greater of these is poverty.

SUMMARY

1. The syndrome of malignant malnutrition as seen in Africans in Durban is described.

2. The essential characteristics are anorexia, wasting, fatty infiltration of the liver and oedema associated with hypo-proteinemia at some stage of the disease.

3. An atrophic stage of the disease is described.

4. The characteristic lesions of the skin and mucous membranes and the associated diarrhoea are considered as manisfestations of complicating deficiency states, mainly due to an associated deficiency in the members of the vitamin B complex.

5. A moderate normocytic, normochromic anaemia is a constant finding.

6. The disease is one of infants and young children, and is related to a diet low in calories and protein and consisting chiefly of carbohydrate in the form of maize and Kaffir corn.

7. Complicating tropical and parasitic diseases were not found.

8. The serum protein concentration was reduced in all the cases.

9. The reduction in serum protein concentration was mainly due to a reduction in the albumin fraction.

10. Albuminuria was not present.

11. Post mortem examinations of the four fatal cases showed enlargement of the liver in three cases and a marked fatty infiltration of the liver in all four.

12. Treatment with a very high protein diet in the form of a soured skim milk formula caused an immediate rise in the serum protein level from a mean initial level of 4.4 gm. per 100 ml. to a mean level of 7.0 gm. per 100 ml. by the 16th to 20th day of treatment associated with a marked diuresis, disappearance of oedema and good clinical improvement.

13. The death rate of the series of 36 consecutive cases was 11% and for the six atrophic cases 33%, but if cases dying within 24 hours of admission are excluded the rates fall to 2.8% and zero respectively.

We wish to thank Dr. Allan Taylor (Medical Superintendent) for permission to publish this paper; Dr. I. Gordon (Senior Government Pathologist) for technical facilities; the residents, Dr. Mayat, Dr. Penny and Dr. Hendrikse for their help with the cases and records and the Sister and Nurses of the Children's Wards for their loyal co-operation and help with these difficult cases.

REFERENCES

Altmann, A. and Murray, J. F. (1948): S. Af. J. Med. Sci., **13,** 91.
Altmann, A. (1948): Clin. Proc., **7,** 32.
Czerny, A. and Keler, A. (1928): *Des Kindes Ernahrung, Ernahrungs-Storungen* and *Ernahrungsstherapie,* 2nd ed. Leipzig.
Gil, A. C. (1934): Rev. Med. Yucatan, **17,** 467 (*quoted by* Waterlow).
Gillman, T., Gillman, J., Inglis, J., Friedlander, L. and Hammon, E. (1944): Nature, **154,** 210.
Gillman, T. and Gillman, J. (1945, a): Arch. Path., **40,** 239.
Gillman, T. and Gillman, J. (1945, b): Arch. Int. Med., **76,** 63.
Gillman, T. and Gillman J. (1945, c): Nature, **155,** 634.
Greenberg (1924): J. Biol. Chem., **82,** 545.
McFarlane, A. L. and Branday, W. J. (1945): Brit. Med. J., **1,** 838.
Nicholls, L. (1946): *Quoted by* Waterlow.
Platt, B. S. (1945): Brit. Med. Bull., **3,** 179.
Rossiter, R. J. (1946): Nature, **158,** 304.
Trowell, H. C. (1937): Arch. Dis. Child., **12,** 193.
Trowell, H. C. (1949): Trans. Roy. Soc. Trop. Med. Hyg., **42,** 418.
Ubico, C. E. and Klee, G. A. (1938): Memoria. Dec. V. Congreso. Medico. Centroamericano, San. Salvador (1942): p. 543 (*quoted by* Waterlow).
Walters, J. H., Rossiter, R. J. and Lehmann, H. (1947): Lancet, **1,** 205, 244.
Waterlow, J. C. (1948): Med. Res. Council Special Report Series No. 263.
Williams, C. D. (1933): Arch. Dis. Child., **8,** 423.

NEW PREPARATIONS AND APPLIANCES

GRAMOLETS—SCHERING

Gramicidin is a pure antibiotic substance, the active principle of tyrothricin. Now available to the medical profession for the first time, *Gramicidin* is presented in stable form free from tissue-irritating substances. As it is the active principle of tyrothricin, it is naturally more potent by weight than tyrothricin itself in the topical treatment of gram-positive infections.

Gramolets, Schering's lozenges, contain as an effective antibiotic for the control of gram-positive organisms, 0.25 mg. Gramicidin; furthermore, each pleasantly flavoured *Gramolet* contains 5 mg. of benzocaine for palliative action. The base of *Gramolets* is slow-dissolving and allows the Gramicidin to have a prolonged action. *Gramolets* contain an agent which renders the Gramicidin soluble and aids it in contacting susceptible surface organisms. Gramicidin activity is not depressed by healthy or diseased tissues nor by saliva or pus in its selective antibacterial action.

Gramolets are indicated for reducing the number of gramicidin-susceptible surface organisms in minor mouth and throat infections, and prophylactically reducing the susceptible 'secondary invaders' on the mouth and throat surfaces in the common cold. Dental surgeons and physicians will find *Gramolets* useful prophylactically, pre- and post-operatively in minor and oral surgery such as dental extractions and tonsillectomies.

Dosage for adults and children: One *Gramolet* dissolved slowly in the mouth, every three hours, or as directed by the physician. No more than eight should be used in 24 hours, except on medical advice.

Gramolets are manufactured in the Union of South Africa by Scherag (Pty.) Limited, Johannesburg, for and under the formula and technical supervision of Schering Corporation, Bloomfield, N.J.

Gramolets are available in tubes of 12 troches.

QUESTIONS ANSWERED

PREGNANCY AND RH INCOMPATIBILITY

Q. The mother is group O, Rh negative; the father is group O, homozygous Rh positive (type Rh_1 Rh_2 (CDe/cDE)); the only child is group O, Rh positive (type Rh_2 (cDE/cde)).

The second pregnancy proceeded normally, antibodies developing in low titre at the 26th week. The titre gradually rose until the 35th week when they were 1 : 512 in serum-albumin. No blocking antibodies ever developed. At the 36th week the titre doubled, but induction of labour was not done. A few days later the child died *in utero*.

The patient has been told that in all future pregnancies the child will be affected and may die *in utero*, or if born alive will require exsanguination-transfusion. If this is correct, can anything be done to prevent an antibody response? Is hapten of any use and if so can it be obtained? Does vitamin K and progesterone, administered throughout pregnancy, prevent antibodies from passing through the placenta? If treatment will not prevent foetal damage, should the patient be allowed to fall pregnant again?

A. The father being homozygous Rh positive, the foetuses of all future pregnancies resulting from this mating will be Rh positive, of the genotypes R_1r (CDe/cde) or R_2r (cDE/cde). The father's genotype should be confirmed by phenotyping his parents and siblings since there is a small chance that his true genotype may be R_2r' (cDE/Cde), in which case he is heterozygous to the Rh_0 (D) factor.

Assuming that the father's genotype is confirmed as R_1 R_2 (CDe/cDE) it is probably inevitable that all future pregnancies will stimulate antibody formation with affection of the foetus to a lesser or greater extent. However, it does not necessarily follow that each succeeding pregnancy will be progressively worse affected, nor that the foetuses will all die *in utero*.

The maternal Rh antibody titre is not a reliable guide to the severity of the affection of the foetus, although the appearance of true blocking antibodies should always be taken as being of grave prognostic significance. More important is the time of first appearance of the antibody or, in the case of carrying over of antibodies from a previous pregnancy, the date of the earliest detectable rise in titre. A useful working rule is, if possible, to avoid having the foetus exposed to the action of maternal antibody for more than 10 weeks by inducing labour at the appropriate time. The risks from prematurity in an affected infant are so great that induction should not be performed more than three or at the outside four weeks before the normally expected date of delivery.

Despite the unavoidable risks of intra-uterine death, replacement transfusion has altered the outlook so dramatically for infants born alive with the disease, that it is doubtful whether a physician is now ever justified in prohibiting future pregnancies.

Up to the present no means have been discovered of counteracting or inhibiting the effects of antibody formation, or of reducing or preventing the facility with which they filter through the placenta into the foetal circulation. Rh hapten, which by definition should be a substance capable of neutralizing Rh antibodies specifically without at the same time having any antigenic action, has been discredited completely.

Artificial insemination (using an Rh negative donor) may provide a different solution to this problem. The medico-legal position is by no means clear, but there can be no biological objections to such a procedure.

ASSOCIATION NEWS : VERENIGINGSNUUS

THE INTERNATIONAL CONGRESS ON CHEST DISEASES

FROM A SPECIAL CORRESPONDENT

The First International Congress on Chest Diseases, under the direction of the American College of Chest Physicians and the auspices of the Italian Government, has been an outstanding success. As a gathering of national representatives it was unique, since almost every country sent representatives—even the U.S.S.R. mustered a party of four. South Africa's delegation consisted officially of seven but only four survived the heat and tension of the five-day session.

Outstanding personalities were Sir Alexander Fleming (discoverer of penicillin) who received the College's gold medal and many floral and verbal bouquets; Prof. Lopo de Carvalho, who had been to the Lourenço Marques Congress in 1938; Prof. Etienne Bernard, Secretary of the International Union against Tuberculosis: Dr. Lehmann, discoverer of PAS, from Sweden; and Professor Morelli, the organizer and founder of the great Carlo Forlanini Institute, a Sanatorium of 3,000 beds, where the Congress met.

Professor Emil Brauer of Hamburg attended the social functions and in company with other great men received much homage and a bombardment by press photographers.

Papers were presented simultaneously in two halls and four languages were used—English, Italian, French and Spanish—while the Russians provided full French translations of their somewhat out-of-date (10-15 years) contributions. Summaries in alternative languages to that of the speaker were thrown on the screen by lantern, but this was far from satisfactory, being too condensed, and not encouraging discussion. Two very efficient lady translators co-operated at all functions, thus lengthening the proceedings considerably.

As usual, for the seasoned attendant at Congresses, the usual old papers (hobby-horses) were trotted out in new guise, but there was also much new and inspiring work. The Portuguese school and the Italians contributed several new items on pulmonary circulation and pathology in relation to angiopneumography and angiocardiography. The French announced new methods of overcoming the shortcomings of streptomycin. At least 70% of the papers dealt with non-tuberculous disease.

The difficulty of limiting Spanish, Italian and French contributors to the regulation 15 minutes was usually countered by a torrential flow of words to beat the clock, conveying the impression of inability to summarize and to eliminate 75% of useless verbiage.

For many the scientific success of the congress depended on a knowledge of the Latin languages, but the spirit of cordiality and fraternity in a common cause was unmistakably present at all sessions and functions.

The social functions, commencing with a long opening session (2½ hours) at the Barberini Palace under a bombardment of electric arc lights and cameras from all directions, included an evening function at the Capitol by the Mayor of Rome, and concluded with a banquet and dance at the premier hotel in Rome. These sessions were very popular and together with outings into the country—Tivoli and the Alban Hills, etc., provided the opportunity of cementing old friendships and making new ones. The Pope received members and fellows of the College in audience and addressed each personally.

Rome was crowded, as was to be expected during the Holy Year and transport conditions were difficult, but the Congress organization provided free transport to and from the distant location.

Altogether this first effort by the American College was a distinct success. Counting its members amongst 62 countries, and representing practising physicians and surgeons as against purely Government nominees of the World Health Organization, its leavening influence in the cause of peace and understanding amongst humanity is definitely much greater.

Dr. D. P. Marais, Regent of the International College of Surgeons for South Africa, was co-chairman of one session with Professor Oekonomopoulos of Athens. Dr. Marais, Dr. T. Schrire and Dr. H. O. Hoïmeyr contributed as 'discussors' by invitation to various papers which were presented.

PASSING EVENTS

Messrs. Maybaker (S.A.) (Pty.) Ltd. announce that their Port Elizabeth Warehouse and Factory will be closed for the annual holidays, from Monday, 11 December until Monday, 1 January 1951.

Emergency orders only will be executed after 8 December 1950.

* * * *

Dr. C. Merskey, M.D. (Cape Town), M.R.C.P. (London) has recently returned after two years' post-graduate study overseas where he devoted his main interest to haematology.

Dr. Merskey has commenced practice as a specialist physician at National Mutual Buildings, Church Square, Cape Town. Telephone:—Rooms: 2-9275. Residence: 2-1471.

* * * *

Congratulations to Dr. and Mrs. H. Meyer on their recent marriage in Boksburg.

* * * *

Congratulations to Dr. and Mrs. Francois le Roux on the birth of a daughter, at George, C.P.

* * * *

Dr. Jean Walker has commenced practice as a specialist dermatologist at 827 Groote Kerk Buildings, Church Square, Cape Town. Telephones:—Rooms: 2-1987; Residence: 7-2064.

REVIEWS OF BOOKS

MEDICAL PROGRESS 1950

Medical Progress 1950. Editor-in-chief, Rt. Hon. Lord Horder, *G.C.V.O.*, M.D., B.Sc., F.R.C.P. (Pp. 524 + v. 37s. 6d.) Butterworth & Co. (Africa) Limited, 1 Lincoln's Court, Masonic Grove, Durban.

Contents: *Part I: Critical Surveys.* 1. Medicine. 2. Surgery. 3. Obstetrics and Gynaecology. 4. Paediatrics. 5. Gastro-Enterology. 6. Bacteriology. 7. Endogrinology. 8. Acute Infectious Diseases. 9. Chest Diseases. 10. Neurology. 11. Tropical Medicine. 12. Urology. 13. Venereal Diseases. 14. Anaesthesia and Analgesia. 15. Public Health. *Part II: Drugs.* 16. Recent Developments in Pharmacology and Therapeutics.
Part III: Abstracts. 17. Abdominal Pain and Acute Abdominal Emergencies—Yaws. Index.

The fascinating and important advances in medical research and practice find good coverage in this volume. Particular interest will attach to the abstracts and reviews of the work on the modern treatment of arthritis. The general review on Medicine contains a very succinct survey of essential hypertension, vagotomy, vascular thrombosis and anti-coagulants and venereal diseases.

Apart from general reviews of the work in every major field of medical practice, the extremely excellent abstract service which comprises the greater bulk of the volume, makes this book virtually an encyclopaedia of medical practice for the year.

As is customary, an important part of the volume is devoted to recent developments in pharmacology and therapeutics.

MITRAL STENOSIS

Le Retrecissement Mitral. By R. Lutembacher. (Pp. 304 with 123 figures. Fr. 1,400.) Paris: Masson et Cie. 1950.

As was only to be expected from such a distinguished contributor to the whole field of cardiology, where he has achieved international recognition due to his classical description of the syndrome which bears his name, this monograph is satisfyingly complete.

No facet has been overlooked in the treatment of his subject, which is not surprising, considering that the material is based on over 300 original communications to the literature on this and allied conditions.

Mitral stenosis is dealt with in all its protean manifestations: with normal rhythm; in the presence of congestive failure; associated with arrhythmias, bacterial endocarditis and congenital anomalies.

The book is adequately illustrated, containing many beautiful representations of pathological material, reproductions of phonocardiograms, electrocardiograms, orthodiagrams and X-ray plates. In addition many illustrative case histories are included which serve to drive home the author's points.

For those who require a reference book which deals exhaustively with mitral disease no better reference source is available.

FAINTING

Fainting: Physiological and Psychological Considerations. By George L. Engel, M.D. (Pp. 141 + xii. With 4 figures. 20s.) Illinois: Charles C. Thomas. England (Oxford): Blackwell Scientific Publications, Ltd. 1948.

Contents: 1. Introduction. 2. Fall in Arterial Blood Pressure. 3. Cardiac Standstill. 4. Cerebral Vascular Disorders. 5. Disturbances in Cerebral Metabolism. 6. Hysterical Fainting. 7. Hyperventilation. 8. Cardiac Disease. 9. Fainting during Air Travel. 10. Sudden Death. 11. Incidence and Diagnosis of Syncope. 12. Differential Diagnosis. References. Index.

This is a most interesting monograph in the *American Lectures in Neurology* series. Syncope is exhaustively considered in relation to the pathological processes of which it may be a symptom. The various types of fainting are studied in the light of recent physiological discoveries and psychological approaches.

Fainting is defined as a sudden, brief cessation of those vital functions which control consciousness, muscle strength and the upright posture: but because loss of consciousness is often preceded by light-headedness, giddiness, faintness and weakness, these symptoms also receive attention.

Three basic mechanisms are submitted as underlying all types of syncope: altered cerebral metabolism due to circulatory disturbances; altered cerebral metabolism due to metabolic factors and psychological mechanisms not involving disturbance in circulation or cerebral metabolism. It is shown that disorders of the heart action account for only a small proportion of syncopal attacks. The most common cause of fainting is a fall in arterial blood pressure, due either to vasodepressor mechanisms which are closely related to fear, or to orthostatic hypotension.

This work contains many important contributions to the understanding of syncope, set out with clarity and authority. Illustrative cases are described, and of particular interest are the electroencephalographic and cardiographic records; much experimental ingenuity must have been exercised to obtain them.

EYE SURGERY

Eye Surgery. By H. B. Stallard, *M.B.E.*, M.D. Cantab.), F.R.C.S. (Eng.). (Pp. 667 + xiii. With 550 illustrations. Second revised edition. 52s. 6d.) Bristol: John Wright and Sons Limited, 1950.

Contents: 1. Introductory. 2. Anaesthesia and Analgesia for Eye Operations. 3. The Eyelids and Reconstructive (Plastic) Surgery. 4. The Lacrimal Apparatus. 5. The Extra-Ocular Muscles: Strabismus and Heterophoria. 6. The Conjunctiva Cornea and Anterior Chamber, and Sclera. 7. The Iris. 8. The Lens. 9. Glaucoma. 10. The Retina, Choroid, and Vitreous. 11. Traumatic Surgery, Civil and Military. 12. The Orbit. Index.

The second edition of this now almost standard work reflects the continued care and painstaking attention to minutiae of the surgical perfectionist. In essence a textbook, it nevertheless bears the stamp of the individualism of its author.

A large part of the text is devoted to the reconstructive surgery of the lids and ocular adnexae for which Mr. Stallard has always shown a gifted predilection. The almost negligible points of criticism one might make in this otherwise practical book are of one or two chapters where somewhat heroic (and perhaps to the more conservative of us) slightly improper procedures are advocated. For example, there is a short chapter on the radiotherapeutic treatment of malignant melanoma of the choroid, and another on the (attempted) surgical removal of the same growth. One feels that these might have been omitted, as it is still generally accepted that early enucleation of the eye with a long optic nerve offers the patient the best hope of survival. Each of us has his surgical foibles and can tolerantly agree or disagree with a talented teacher and colleague. One feels, however, that the yardstick in surgical endeavour should always be the temporary mental substitution of oneself for the patient; and if any one of us should ever fall the unfortunate victim to a malignant melanoma of the choroid, in the words of the well-known radio character: 'What would *you* do, chums?'.

MEDICAL PHOTOGRAPHY

An Introduction to Medical Photography. By Josephine Hunt, S.R.N., S.R.C.N., F.I.B.P., F.R.P.S. (Pp. 243. With 86 illustrations. 30s.) London and New York: Staples Press. (*South African Representative*: Mr. Paul Kosten, P.O. Box 4782, Cape Town.) 1950.

Contents: *List of Illustrations. Foreword. Preface.* 1. The Photographer. 2. The Photographic Department and Equipment. 3. Dark Rooms—Equipment and Technique. 4. The Administration of the Photographic Department. 5. The Patient. 6. Methods of Photographing the Straight-Forward Case. 7. Methods of Photographing the Difficult Case—I. 8. Methods of Photographing the Difficult Case—II. 9. The Reproduction of Radiographs. 10. Photography of Pathological Specimens. Copying. Microcopying. Filmstrip, Reflex Printing, and Lantern Slide Making. 11. Printing by Contact and Projection. 12. Cardiography. Conclusion. Bibliography. Index.

The publication of another book on medical photography is a sign of the times. A photographic department is regarded today as an indispensable part of every well-equipped hospital. A good photograph adds meaning and value to hospital records. In teaching, photography is assuming an ever-increasing importance.

This book does not aim to teach one photography, but is full of very useful and practical advice in the running of a photographic department at a hospital. The chapters dealing with hospital routine and personal relationships with the nursing staff and other departments indicate considerable experience on the part of the author and point the way to run the department in an efficient manner and with a minimum of friction.

The author gives helpful advice about the lay-out and equipment of a photographic department. The cameras and methods of taking the photographs are not dealt with exhaustively, but represent the author's personal approach and preferences. While, for some reason best known to the author, there is an account of cardiography, photomicrography and cinematography are considered beyond the scope of this book.

Though this book does not contain all the answers, those starting a photographic department at a hospital will find much valuable advice and guidance in its 243 pages.

THE TRIAL OF JESSIE M'LACHLAN

The Trial of Jessie M'Lachlan. Edited by William Roughhead. (Pp. 402 + xi. With 10 illustrations. 3rd ed. 15s.) Edinburgh and London: William Hodge and Company, Limited. 1950.

Contents: 1. Introduction. 2. Table of Dates. 3. The Trial. 4. First Day, Wednesday, 17 September 1862. 5. Second Day, Thursday, 18 September 1862. 6. Third Day, Friday, 19 September 1862. 7. Fourth Day, Saturday, 20 September 1862. 8. Appendices.

Jessie M'Lachlan was tried 88 years ago and convicted of a murder which she may not have committed. Responsible opinion has placed her guilt no higher than as a possible accessory and perhaps even an unwitting one. It was alleged that she did to death with an iron chopper her friend, a domestic servant, working for her former employer. The motive was alleged to be the theft of a few paltry articles and there is a considerable body of evidence that the murder may actually have been committed by an old man of some 80 years who was living in the house and who had made improper advances to the deceased.

Unfortunately for the accused, her inveterate capacity for lying served only to make her own case more difficult and possibly to allow the real murderer to go scot-free.

The trial of Jessie M'Lachlan is remarkable in very many ways. The statement made by the accused immediately after her conviction was treated with the greatest contempt by the presiding judge (Lord Deas); but it nevertheless precipitated a nation-wide agitation which forced the Government to appoint a Crown Commissioner to hear new evidence. The uproar resulted in commutation of the death sentence to penal servitude for life.

For the medical practitioner the verbatim record of this trial holds a particular interest. The evidence given by Dr. MacLeod in his autopsy report called forth from Lord Deas the observation that, as a police surgeon, Dr. MacLeod entered in his report matter which was not suitable to such a medical report (p. 119). Lord Deas made this criticism of the medical evidence on more than one occasion and although the case is an old one, a careful study of the medical evidence forms an excellent guide to the modern district surgeon about how not to record his autopsy findings and how not to give expert evidence.

Dr. McLeod was unwise enought to set out in his report the opinion that the deceased had been murdered and that with extreme ferocity (p. 121). This, of course, was a most improper conclusion to advance as it was for the Court to find whether the deceased had been murdered. Furthermore, Dr. MacLeod, in inferring from the wounds he observed that they had been inflicted with extreme ferocity, revealed talents which all other pathologists lack. Needless to say, there were other medical witnesses who disagreed very strongly with these opinions and the result is an entertaining demonstration of the problems of giving expert testimony.

To-day it is quite incredible that a medical practitioner should have considered it his functon to give expert evidence on the identity of footprints. It is also a chastening reflection that proof of the contamination of an exhibit by blood depended on the allegedly incontrovertible scientific fact that incineration of the material under test produced 'the peculiar odour of burnt blood' and the further highly logical deduction that the odour of burnt blood is highly characteristic of blood.

An interesting medico-legal problem was presented by the number and the situation of the wounds inflicted on the deceased. Some of the medical witnesses were most dogmatic that the deceased could not have received the blows if she was standing. It is a common-place in analysing this kind of situation that everything depends upon the relative positions of the assailant and the victim. This contrary view was very well brought out by other medical witnesses. The study of this evidence is extremely valuable to those who may find themselves in the position of having to give expert testimony in cases of assault, whether fatal or otherwise.

Another interesting experience is that endured by one of the witnesses for the Crown, a Dr. Fleming. He was emphatic that the marks of blood he saw on the kitchen door and on

the floor were certain evidence that a struggle had taken place. It is doubtful whether this is an expert opinion which Dr. Fleming should have proffered, but in any event his dogmatism made him very easy meat for the skilful cross-examination which can be read in detail on pp. 360-361. An expert witness ends up in a thoroughly deflated condition when he has to admit that his dogmatic assertion that a struggle took place depends upon the appearance of blood smears equally consistent with the conjecture that there was no struggle at all.

Medical and lay reader alike will find fascinating the record of this extraordinary trial about which Sir Archibald Alison wrote with pride of the part he had played in delivering Jessie M'Lachlan from the sentence of death. He went so far as to state that Jessie M'Lachlan's execution would have been a judicial murder (p. 60).

Modern Trends in Obstetrics and Gynaecology

Modern Trends in Obstetrics and Gynaecology. Edited by Kenneth Bowes, M.D., M.S. (Lond.), M.B., Ch.B. (Liverpool), F.R.C.S. (Pp. 778 + xi. With 139 illustrations. 70s.) Butterworth & Company (Africa) Limited, 1 Lincoln's Court, Masonic Grove, Durban. 1950.

Contents: Introduction. 1. The Anatomy of the Bony Pelvis and the Pelvic Floor. 2. The Vascular Anatomy of the Adult Human Uterus. 3. Sex and Intersexuality. 4. Statistical and Genetical Problems. 5. Psychological Factors in Obstetrics and Gynaecology. 6. Aspects of Present Knowledge of the Pituitary Trophins and Steroid Hormones. 7. Radio-Isotopes in Research. 8. Phases of Human Development. 9. The Physiology of the Placenta. 10. Aspects of Foetal Physiology. 11. Abnormal Developmental and Foetal Death. 12. The Rhesus Factor in Pregnancy. 13. The Presentation of the Foetus. 14. Social Factors in Obstetrics. 15. The Study of Uterine Contractions by Hysterography. 16. Abnormalities of Uterine Action in Labour. 17. The Diagnosis and Treatment of Disproportion. 18. The Toxaemias of Pregnancy. 19. Anuria in Pregnancy. 20. Haemorrhage Associated with Pregnancy and Childbirth. 21. Shock in Obstetrics. 22. Infections of the Genital Tract following Childbirth and Abortion. 23. Heart Disease and Pregnancy. 24. Pregnancy and Diabetes Mellitus. 25. Tuberculosis and Pregnancy. 26. The Anaemias of Pregnancy. 27. Anaesthesia in Obstetrics and Gynaecology. 28. Radiology in Obstetrics and Gynaecology. 29. Operative Obstetrics. 30. Lactational Physiology. 31. The Cytology of the Vaginal Smear. 32. Pre-Malignant Lesions of the Cervix Uteri. 33. The Cytology of the Uterine Epithelia. 34. Latent Tuberculosis of the Endometrium. 35. The Study of Tubal Motility by the Kymograph. 36. Fertility and Infertility. 37. The Pathology and Clinical Characteristics of Ovarian Neoplasma. 38. Endometriosis. 39. The Treatment of Abortion. 40. Endocrine Therapy. 41. Radiotherapy in Gynaecology. 42. Venous Thrombosis and its Treatment. 43. The Operative Treatment of Genital Prolapse. 44. Hysterectomy in Non-Malignant Conditions. 45. The Surgery of Uterine Malignant Disease. 46. The Surgery of Malignant Disease of the Vulva. 47. Gynaecological Urology. 48. Conservative Gynaecological Surgery. 49. Pelvic Sympathectomy in the Treatment of Dysmenorrhoea. 50. The Law in Relation to Obstetrics and Gynaecology. Index.

This is an outstanding publication on obstetrics and gynaecology and a landmark in British literature on the subject. Never before has a work like this been compiled in Britain. There have been books on *Recent Advances* by one or two authors, annual reviews of the literature, and textbooks by a series of men from the same school; but here we have a book on *Modern Trends in Obstetrics and Gynaecology* by no less than 60 of the leading men in the country, each writing on the aspect of the specialty in which he has done particular research or has had particular experience. Thus the section on Endocrinology is done by Bishop whose reputation in this branch is well known; Disproportion by Chassoir Moir of Oxford; Ante-Partum and Post-Partum haemorrhage by Macafee of Belfast; Toxaemias by Kellar of Edinburgh; Endometriosis by MacLeod; the Rh factor by Mollison—to mention but a few of Britain's leading scientists and gynaecologists who have contributed to the work.

The editor has not limited his choice of authorship to the British Isles. Thus Heyman of Stockholm, probably the world's leading gynaecological radiotherapist, has written the chapter on Cancers; the famous pelvic anatomist from Australia, Maguire, has given a chapter on the Pelvis; likewise there are contributions from Canada, New Zealand, and Denmark, and a few from the United States.

The ancillary sciences to obstetrics and gynaecology have been included and handled on the same high standard. There are sections on *Social Factors in Obstetrics, Radio-Isotopes in Research, Psychological Factors in Obstetrics and Gynaecology,* and *Statistical and Genetical Problems.*

No better tribute could be paid to the book than to mention the names of these authors and their topics. Nearly every one is a world authority on the subject of which he has written.

Though the volume is a big one, a possible criticism is that certain important subjects with modern advances have been omitted, e.g. one would have liked to have seen a chapter by Sheehan on Obstetric Pathology; Modern Chemotherapy as applied to Obstetrics and Gynaecology; Pruritus Vulvae (with recent views on its aetiology) and Prematurity. Had these and a few other gaps been filled, this outstanding volume would have been complete.

It is to be hoped that this type of work will make a regular appearance in the future. The reception that the present volume will receive will clearly indicate the need for such a feature in British literature on gynaecology and obstetrics.

Cytological Diagnosis of Cancer

The Cytologic Diagnosis of Cancer. By the Staff of the Vincent Memorial Laboratory of the Vincent Memorial Hospital. (Pp. 229 with 153 figures. 55s. 3d. or $6.50.) Philadelphia: W. B. Saunders Company. 1950.

Contents: 1. Normal Cells from Cervical and Vaginal Squamous Epithelium. 2. Normal Cells from Columnar Epithelium of Endocervix and Endometrium. 3. Cells not originating from Epithelium of Female Genital Tract. 4. Squamous Cell Carcinoma of the Cervix. 5. Adenocarcinoma of the Endometrium. 6. Adeno-acanthoma of Uterus. 7. Other Tumors of the Female Genital Tract. 8. Radiation Changes in Normal and Malignant Cells of the Vaginal Secretion. 9. Cells of Squamous Epithelium of Respiratory Tract. 10. The Columnar Epithelium of Respiratory Tract. 11. Cells of Non-epithelial Origin in Sputum. 12. Squamous Cell Carcinoma of the Lung. 13. Other Types of Pulmonary Carcinoma. 14. Normal Columnar Cells from Gastric Mucosa. 15. Carcinoma of the Stomach. 16. Normal Cells of the Urinary Tract. 17. Carcinoma of the Genito-Urinary Tract. 18. Normal Cells of Pleural and Peritoneal Fluid. 19. Malignant Cells in Pleural and Peritoneal Fluid. 20. Technic. Appendix. Bibliography. Index.

This monograph is a comprehensive and authoritative piece of work on the cytological diagnosis of carcinoma and reflects the wide experience of the staff of the Vincent Memorial Laboratory. It is the result of careful and detailed study of the subject over a period of many years, commencing with the study of vaginal smears for carcinoma, which has proved of such value. The work has extended to smears of sputum, bronchial secretions, gastric secretions and serous fluids, which are all fully described in the text.

The emphasis of the book is naturally on cell detail, on which a wealth of information is given. The authors have taken great trouble to present clearly the features of smears of normal tissue cells, variations within the normal limits and their comparison with malignant cells from the same organ. Infinite care, too, has been taken with the very beautiful and very helpful illustrations which appear as low- and high-power photomicrographs, often accompanied by drawings to indicate precisely the cells under discussion. Several of the illustrations appear in colour. There are innumerable tips for the diagnosis of the cell type and warnings of the pitfalls. Of particular value is the chapter on radiation changes in normal and malignant cells.

The excellent bibliography contains over 200 references. This book should also prove a valuable reference book to all pathologists.

The publisher, too, is to be congratulated on the artistic beauty of the book.

A Cancer Atlas

Atlas of Tumor Pathology. Section II—Fascicle 6. Tumors of the Peripheral Nervous System. By Arthur Purdy Stout, M.D. (Pp. 57 with 56 figures. 60 cents.) Published by the Armed Forces Institute of Pathology under the auspices of the Subcommittee on Oncology of the Committee on Pathology of the National Research Council, Washington, D.C. 1949.

Contents: 1. Tumours of the Peripheral Nerves. 2. Tumours of Sympathetic Ganglia. 3. Tumours of Paraganglionic Cells. 4. Complex Malignant Neoplasms. 5. Neoplasms of Heterotopic Central Nervous System Tissues.

Although this *Atlas of Tumour Pathology* has been prepared for use in connexion with the teaching programme of the United States Armed Forces Institute of Pathology and the activities of other medical units of the National Military

Establishment, the National Institute of Health and other Federal agencies, it will provide a most excellent service in Medical Schools generally and in the day-to-day work of clinical pathologists.

The first section available is Dr. A. P. Stout's magnificent account of the peripheral nervous system. South African readers will be interested to see amongst the illustrations several photomicrographs which photographs which appeared in Dr. Stout's excellent paper in *Clinical Proceedings* in 1946. Each illustration is related to a concise account in the text and this makes the Atlas invaluable to those who are involved daily in diagnostic morphological problems.

The full scope of the work can be seen from the following list of Sections to be published: 1. Tumours of the Skin and Cutaneous Melanomas. 2. Tumours of the Musculo-skeletal System. 3. Tumours of the Cardio-vascular and Hematopoietic Systems. 4. Tumours of the Oral Cavity, Upper Respiratory Tract, Neck and Ear. 5. Tumours of the Lower Respiratory Tract and Thoracic Contents. 6. and 7. Tumours of the Digestive System. 8. Tumours of the Uro-genital System and Adrenal. 9. Tumours of the Female Generative System. 10. Tumours of the Intracranial Structures and Eye. 11. Synonyms and Index.

The various sections will be issued separately in loose-leaf form and this method of publication will enable the Editors to keep the Atlas up to date, constantly improving it.

It will be issued free of charge to laboratories of the Armed Forces and other government agencies and may be obtained at cost from the Government Printing Office, through the American Registry of Pathology, Armed Forces Institute of Pathology, Washington, D.C.

The importance of this project is so great that there can be little doubt that every medical school and every private pathologist will find it essential to have the Atlas available for ready reference.

Tumour Formation

Studies on Tumour Formation. By the late G. W. de P. Nicholson, M.A., M.B., B.Ch. (Pp. 598 + xii. With 183 illustrations. 71s.) Butterworth & Company (Africa) Limited, 1 Lincoln's Court, Masonic Grove, Durban. 1950.

Contents: 1. Introduction. 2. Tissue Malformations: Anomalies of Bulk of Differentiation. 3. Tissue Malformations: Anomalies of Position and of Blending. 4. Acquired Tissue Malformations. 5. The Importance of Congenital Malformations in Tumour Formation. 6. The Hypernephromata, or Strumae Suprarenals Aberratae. 7. The Heterotopic Tumours. 8. The Mixed Tumours. 9. The Mixed Tumours (Continued). 10. The Teratomata. 11. Causation: Reaction and Environment. 12. Method in Oncology—The Organism and the Tumour. 13. Somatic Development and Tumour Formation. 14. Kidney in a Teratoma. 15. A Foetiform Ovarian Teratoma. 16. Foetiform Ovarian Teratomata (A review of the literature). 17. An Abdominal Foetiform Teratoma. 18. A Sacro-Coccygeal Teratoma with Three Metacarpal Bones and Digits. 19. Ovarian Goitre. 20. Induction and Determination.

This book has been published as a companion volume to Willis' *Pathology of Tumours* by Butterworth & Co. Ltd., and is a joy to read.

G. W. Nicholson was well known as Professor of Pathology at Guy's Hospital, London, and during the years 1922 and 1938 he published, in the Guy's Hospital Reports, a series of 20 *Tumour Studies*. These studies have now been reprinted in their original form in this handsome volume, which constitutes a treasure to be read and re-read for sheer delight.

The studies are a classic of tumour research, covering a wide variety of different subjects. They contain a wealth of original observations recorded with the most meticulous accuracy and made attractive by the personality of the author and his almost conversational style. One is amazed by the quality of the numerous illustrations of histological preparations, everyone of which was drawn in freehand by Nicholson himself.

Some of the subjects, e.g. the general structure of tumours, malformations, heterotopias, mixed tumours and teratomas, are dealt with in a way that reveals an immense knowledge not only of pathology but of embryology and biology in general, written at a time when it was so urgent to rectify the misconceptions of Cohnheim, Grawitz and Wilms, and to interpret teratology on the lines of the work of the experimental biologists. These were the circumstances in which the papers were published and although in 1948 he had intended to amend some of the earlier papers in the light of later experience, Professor Nicholson's death came before he could start.

To his former colleagues at Guy's Hospital, who have chosen this way to perpetuate his memory, pathologists all over the world will indeed be grateful.

History of Medicine

Essays in the History of Medicine. By George Gask. (Pp. 209 + viii. Fully illustrated. 34s. 6d.) Butterworth & Company (Africa) Limited, 1 Lincoln's Court, Masonic Grove, Durban. 1950.

Contents: 1. Early Medical Schools. 2. Vicary's Predecessors. 3. The Medical Staff of King Edward the Third. 4. The Medical Services of Henry the Fifth's Campaign of the Somme in 1415. 5. A Contribution to the History of the Care of the Sick and Wounded during Marlborough's March to the Danube in 1704, and at the Battle of Blenheim. 6. John Hunter in the Campaign in Portugal, 1762-3. 7. Lettsomian Lectures: 1. Historical Sketch of the Methods of Treating Wounds of the Chest in War from A.D. 1300 to 1900. 2. The Treatment of Wounds of the Thorax during the War of 1914-1919. 3. The Present Position of Surgery with Reference to Diseases of the Thorax. 8. Changing Surgery.

The history of medicine offers a subject of enduring interest to those who practise it and the late Dr. Gask's volume is an important and valuable contribution to this field.

Of particular interest is the considerable amount of material made available to the reader from primary sources. The volume consists of a collection of essays and addresses and covers ancient as well as more modern times.

The considerable critical capacity of the author is revealed in his masterly dissection of the problem of the origin of Hippocratic medicine and the clear demonstration that it is to be divorced from the non-scientific magic and mystery of the temple-medicine cults which preceded it. The author also effectively disposes of the myth that Hippocrates based his theory and his practice on what went on in the Aesculapium in Cos.

The history of chest wounds is a very comprehensive one and South African practitioners will be particularly interested in the passage (on page 157) in which Dr. Gask states that the mortality from chest wounds in the South African war was the lowest on record and that owing to the character of the weapons used (the high-velocity low-calibre rifle) the wounds inflicted were generally not very severe and did not commonly require active surgical treatment.

Elements of Genetics

Elements of Genetics. By Edward C. Colin, Ph.D. (Pp. 418 + xiii. With 93 Figures. $3.50. Second edition.) Philadelphia: The Blakiston Company.

Contents: 1. Mendel: Student, Priest, Teacher, Investigator. 2. Dominance and the Law of Segregation. 3. Dihybrids: The Law of Independent Assortment. 4. Chromosomes and Mendel's Laws. 5. The Factor Principle: Action and Interaction of Genes. 6. The Rediscovery of Mendel's Work. 7 Linkage and Crossing-Over. 8. Heredity in Man, I. 9. Heredity in Man, II. 10. Sex Determination and Sex Differentiation. 11. Sex-Linked Heredity. 12. Heredity and Enviroment. 13. The Gene and Mutation. 14. Inbreeding and Crossbreeding. 15. Heredity and Evolution. 16 Improvement of the Human Species (Eugenics). Glossary. Index. Laboratory Exercises

This is one of the most generally useful accounts of biological inheritance written in a manner which the medical practitioner as well as the medical student will find particularly attractive.

There is a considerable amount of material on human heredity and the account of the blood groups is very adequate. The need to pay more attention, in medical education, to the study of heredity in man is becoming recognized increasingly by our medical educationists and chapters 8 and 9 form a particularly concise and useful summary of the situations that the medical student should be concerned about.

The book is produced with distinction and the excellent illustrations as well as the clearly written text communicate to the reader much of the excitement associated with biological exploration and discovery.

Dr. Colin's book is a most adequate traveller's guide to the fascinating journey in the field of human heredity which scientific endeavour has now made possible.

ART OF THE BLIND

Psychology and Art of the Blind. By G. Révész. Translated from the German by Dr. H. A. Wolff (Pp. 338 + xiv. With 105 illustrations. 42s.) London, New York, Toronto: Longmans, Green & Company. 1950.

Contents: *Part I.* Space Psychology and Haptic Space. 1. The Problems of Space Psychology. 2. Fundamental Problems of Haptics. *Part II.* The Aesthetics of Form and the Art of the Blind. 1. The Aesthetic Experience in Haptics. 2. The Creative Activity of the Blind.

This work is a valuable contribution to research in the field of haptics in its special relation to the psychology of the blind.

The author approaches his subject from a new angle and presents his arguments clearly. He emphasizes that the problems of form in the field of haptics are completely different from those in the field of optics, stating that: 'Haptics is completely independent of the sense of vision and creates its world through its own activity and its own laws.'

This distinction was not fully appreciated in the past and gave rise to the erroneous conclusion that 'the sense of touch is governed by laws and principles originating in the field of visual perception.'

One cannot sufficiently emphasize the importance of this work to all teachers of the blind, presenting as it does a sound theory on which to base the development of their artistic abilities by defining their scope more scientifically than has been done in the past.

PROFESSIONAL NURSING

Progressive Professional Nursing. By Mona E. Grey, S.R.N., S.C.M. (Pp. 104. 6s.) Edinburgh: E. & S. Livingstone Limited. 1950.

Contents: 1. Great Britain. 2. Ireland. 3. The Public Health Field of Nursing. 4. The Profession.

Miss Grey has managed in the small space of this little book to give a very clear picture of nursing and all that it means in the United Kingdom of the present day. While there is something of the history of nursing of the pre-Nightingale days, it is as well to remember that it is only 90 years since that great lady founded the first endowed School of Nursing at St. Thomas' Hospital. A great deal has happened in nursing since that time and Miss Grey tells the story in a concise and interesting way.

With the nationalization of Health Services in the United Kingdom, the place of nursing in the general scheme is clearly set out.

ANXIETY IN PREGNANCY

Anxiety in Pregnancy and Childbirth. By Henriette R. Klein, M.D., Howard W. Potter, M.D., and Ruth B. Dyk, M.S. (Pp. 111 + ix. $2.75.) New York: Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers.

Contents: *Preface. Acknowledgments.* 1. Description of Study. 2. Attitudes and Reactions Towards Conception and Pregnancy. 3. Anxieties: Superstitions and Misconceptions. 4. Psychosomatic Relationships in Pregnancy and Childbirth. 5. Summary. Bibliography. Appendices. Case Histories.

This volume, beautifully bound and equally magnificently printed, is a little disappointing in its contents. Although the subject matter, viz. the investigation of the specific emotional components of the physiological phenomenon of childbearing, is one of interest, the ground covered is very limited. The one factor common to all the women investigated is their primiparous state. For the rest many of them differ widely in race, religion, social status, etc.

As only 27 patients were investigated in all, it is clear that the conclusions drawn must be of a very cautious nature and are therefore not as useful as one hoped on first opening this very fine-looking book.

The authors themselves state that this is merely a pilot study but feel that 'it does bring into focus significant points which need continued scrutiny'.

Several appendices and case histories will be found at the end of the volume for the guidance of those interested in pursuing the topic further.

VIRAL DISEASES

The Pathogenesis and Pathology of Viral Diseases. Edited by John G. Kidd (Symposium held at the New York Academy of Medicine, December 1948). (Pp. 235 with tables and plates. $5.00.) New York: Columbia University Press. 1950.

Contents: 1. Introduction. 2. The Spread of Viruses from Infected to Susceptible Hosts. 3. The Culture and Effects of Viruses in Chick Embryo Cells. 4. The Relationship of Viruses and Cells, with Particular Reference to the Interference Phenomenon. 5. The Activators of Viruses by Adsorption Co-Factors. 6. The Electron Microscopic Study of Virus Growth 7. Pathology and Pathogenesis of the Cutaneous Lesions of Variola, Vaccinia, Herpes Simplex, Herpes Zoster, and Varicella. 8. Pathogenesis of the Viral Exanthems as Exemplified by Mouse Pox (Infectious Ectromelia) of Mice. 9. Cardiac Lesions Produced by Viruses. 10. Pathology of Yellow Fever. 11. Reaction of the Cells of the Respiratory Tract to Virus Infections. 12. Proliferative Lesions caused by Viruses and Virus-like Agents. 13. The Pathology of Lymphatic Choriomeningitis Virus Infection: A Discussion. 14. The Pathology of Some Viral Encephalitides 15. The Nature and Pathogenesis of Neuronal Changes in Poliomyelitis.

This is the last of three volumes publishing symposia of the Section on Microbiology, held at the New York Academy of Medicine. Like each of its companion volumes, it covers an important field of microbiology. The scope of this volume is, relatively, a wide one. It, nevertheless, summarizes, very effectively, an enormous volume of work on several aspects of viruses. The book is concerned with current virus problems and is therefore primarily of interest to the research worker. For the majority of medical practitioners it is impossible to keep in touch with developments in laboratory research, particularly in the virus field, even if there is the necessary desire to do so. A book such as this affords an outstanding opportunity to learn of the developments in the virus field in concise articles written by well-known authorities.

Many of the chapters are authoritative summaries of work already published elsewhere. For instance, Anderson's chapter on the activation of viruses by absorption co-factors describes briefly the experiments with the T phages of *B. coli*, which show that some strains of phage require co-factors such as tryphophane before specific absorptions to their hosts will occur. Other chapters introduce the results of new investigations. Of particular interest to clinicians will be the report of cardiac lesions produced in rabbits by a number of different viruses. The cardiac pathology depends not only on the presence of the virus, but also on a mechanism which lowers the oxygen supply of the heart during the initial period of infection.

This little book is, to every practitioner, well worth its price and the time spent in reading it.

PAEDIATRICS

Mitchell-Nelson Textbook of Pediatrics. Edited by W. E. Nelson, M.D. (Pp. 1,658 with 426 illustrations, 19 in colour. £5 6s. 3d. or $12.50. 5th ed.) Philadelphia: W. B. Saunders Company. 1950.

Contents: 1. The Field of Pediatrics. 2. Care and Evaluation of Well Children. 3. General Factors in the Care of Sick Children. 4. Prenatal Disturbances. 5. The Newborn Infant. 6. The Premature Infant. 7. Unexpected Sudden Death. 8. Nutritional Disturbances. 9. Miscellaneous Diseases. 10. Malignant Tumors in Early Life. 11. Infectious Diseases. 12. Poisoning from Drugs, Metals and Food. 13. The Digestive System 14. The Respiratory System. 15. The Cardiovascular System. 16. The Spleen. 17. The Lymphatic System. 18. The Thymus Gland. 19. Disturbances of Cellular Lipid Metabolism and related Conditions. 20. The Genito-Urinary System. 21. Psychologic Disorders. 22. Disorders in Language Functions. 23. Convulsive Disorders. 24. The Cerebral Palsies. 25. The Endocrine System. 26. Diabetes Mellitus. 27. Hypoglycemia. 28. The Bones and Joints. 29. The Muscles. 30. Allergic Diseases. 31 The Skin. 32. The Eye. 33. Adolescence. Appendix.

This well-known textbook has enjoyed an assured and deserved popularity in the past. The publication of a new edition within five years of the last, is an index not only of the wide acceptance of the book, but also of the extensive advances in paediatrics during that period. A praiseworthy attempt has been made to cover the entire field of child care in the scope of one volume. This can be no easy task and one must applaud the able editorship of Dr. Nelson. The 63 contributors, all of whom are American, have been chosen as recognized authorities on their particular subjects.

A number of new sections has been added to this edition; many other chapters have been rewritten in keeping with

current knowledge, and one may regard this volume as up-to-date in all respects. Diagrams and illustrations are adequate and a valuable list of references is appended to each section. The general lay-out of the book has been particularly well planned.

One may strongly recommend this as a paediatric reference book to both students and practitioners.

INTRODUCTION TO BIOCHEMISTRY

Hoofdlijnen der Biochemie. By Dr. F. A. Steensma. (Pp. 432. F.16.50.) Amsterdam: Scheltema & Holkema N.V. 1950.

Contents: 1. Eerste Afdeling. 2. Scheikunde Samenstelling van Het Menselijk Lichaam. 3. Scheikundige Functies van Organen en Weefsels. 4. Stofwisselingsprocessen (Metabolisme).

The subject matter of this book has been dealt with under two headings, general descriptive biochemistry and the biochemistry associated with the function of the various organs and tissues of the body.

In the first half of the book an account is given of substances of biochemical interest such as carbohydrates, fats, proteins, vitamins, hormones, pigments, etc. This section includes a short account of vitamin B_{12} and reference is also made to cortisone and to hyaluronidase. Two chapters are devoted to the heterocyclic compounds which include such important substances as the porphyrins and purines and a special chapter deals with the steroids.

The second half includes sections on the composition of blood, sweat, gastric juice, urine, etc., the functions of the kidney and liver and metabolism. Mineral metabolism and trace elements are also adequately dealt with. A short account is finally given of the new important concept of the phosphate bond energy and its relationship to the chemistry of muscular contraction.

The book is simply yet accurately written and all the most recent developments in biochemistry are included. It is an excellent introduction to modern biochemistry and can be strongly recommended to all those who wish to keep abreast of the biochemical times.

RECENT ADVANCES IN PHARMACOLOGY

Recent Advances in Pharmacology. By J. M. Robson, M.D., D.Sc., F.R.S.E., and C. A. Keele, M.D., F.R.C.P. (London). (Pp. 400 + xi. With 46 illustrations. 24s.) London: J. & A. Churchill Limited. 1950.

Contents: 1. Anti-Cholinesterase Drugs. 2. Curare, Mephenesin (Myasesin), and Tetraethylammonium Salts. 3. Anti-Histamine Drugs. 4. Sensitization to Drugs (Drug Allergy). 5. The Steroids of the Gonads and Adrenal Cortex. 6. The Thyroid. 7. Bacterial Chemotherapy. 8. Antimalarial Drugs. 9. Antrycide. 10. Chemotherapy of Malignant Disease. 11. Mutagenic Substances. 12. Analgesic Drugs. 13. The Haemopoietic System. 14. Anticoagulant Drugs. 15. The Mode of Action of Drugs. 16. Methods of Studying the Fate of Drugs in the Body. 17. Evaluation of the Actions of Drugs in Man. 18. The Control of the Body Functions by Chemical Substances. Index.

The vast and growing field of pharmacology has made it difficult even for the specialist to keep pace with the course of events. Numerous drugs have been discovered and great advances made in our knowledge of their actions and uses. The authors of this book have confined themselves to important aspects of the subject likely to be of interest to students and practitioners of medicine, especially emphasizing the actions of drugs and the basis for their use in practice.

The review of the various topics is not exhaustive, but the provision of key references will allow original sources of information and review articles to be consulted. Not much will be found with regard to drugs like the sulphonamides which are no longer in the forefront of development. Due emphasis is, however, given to the mechanisms by which drugs produce their actions, and their therapeutic use.

A valuable feature included is information on the recently introduced techniques for elucidating the actions of drugs. Formulae, tables, diagrams and photographs are abundantly provided throughout, and most statements are documented.

As examples of subjects discussed the following may be cited: anticholinesterase drugs and the newly discovered alkyl phosphates; the neuromuscular blocking action of curare and synthetic curariform drugs, the ganglionic blocking actions of tetraethylammonium salts, and special nervous depressants like mephenesin (Myanesin); evaluation of antihistaminic drugs; the toxic effects of drugs due to overdosage or to abnormal reactions such as sensitization; the steroids; thyroxine and antithyroid substances; the antibiotics, including aureomycin; newer knowledge on the malarial parasite and a survey of antimalarial drugs; the preliminary results with Antrycide in trypanosomiasis in cattle; the substances of value in neoplastic disease; and many other subjects under the headings given in the list of contents.

Practitioners, specialists and research workers will find in this book a most useful account of the latest developments in pharmacology.

AIDS TO SURGICAL ANATOMY

Aids to Surgical Anatomy. By J. S. Baxter, M.D., M.Sc., F.R.C.S.I. (Pp. 203 + vii. 5s. Third edition.) London: Baillière, Tindall & Cox. 1950.

Contents: 1. The Head and Neck. 2. The Upper Extremity. 3. The Thorax. 4. The Abdomen. 5. The Lower Extremity. 6. The Autonomic Nervous System. Index.

For a few shillings the student has here available a portable volume for use in his clinical surgical studies. This new edition has been rewritten to present modern teaching and practice, with a chapter on the autonomic nervous system and the principles on which certain operations on it are based. There are many illustrations.

OBSTETRICS

A Pocket Obstetrics. By Arthur C. H. Bell, M.B., B.S., F.R.C.S., F.R.C.O.G., Hon. M.M.S.A. (Pp. 156 + viii. With 14 illustrations. Second edition. 7s. 6d.) London: J. & A. Churchill Limited. 1950.

Contents: 1. Anatomy. 2. Normal Pregnancy, Labour and Puerperium. 3. Abnormal Pregnancy. 4. Abnormal Labour. 5. Abnormal Puerperium. 6. Obstetric Operations. 7. The Baby.

The practice of obstetrics, both normal and abnormal, is concisely presented in this handy pocket volume. The facts and practical advice follow those laid down in the Queen Charlotte's *Textbook of Obstetrics*. General practitioners and students will find this a useful work for easy revision of the subject.

CORRESPONDENCE

STREPTOMYCIN TREATMENT

To the Editor: I have to inform you that a communication has been received from the Director-General of the World Health Organization, Geneva, Dr. Brock Chisholm, in which a warning is issued against the unrestricted distribution and indiscriminate use of streptomycin in treating tuberculosis.

Attention was drawn to the following facts:—

i. Although new forms of streptomycin have reduced toxicity, the antibiotic must still be considered dangerous from the point of view of toxicity to the patient because of the fact that the emergence of streptomycin-resistant forms of tubercle bacilli constitutes an important public health hazard.

ii. Precise knowledge as to its clinical indications, especially in pulmonary tuberculosis, is still lacking.

iii. The attention of the public may be focussed on this antibiotic to a degree out of all proportion to its value in the overall campaign against tuberculosis.

It will be appreciated if publicity could be given to this matter in the columns of the *S. Af. Medical Journal.*

Secretary for Health.

Department of Health,
P.O. Box 386,
Pretoria.
25 September 1950.